--41. A method of ablating auto-antigen-specific T cells in an auto-immune disease patient comprising the steps of:

removing antigen presenting cells (APCs) from an auto-immune disease patient;

transferring into the APCs a polynucleotide which encodes all or a portion of an auto-antigen to which the patient's antigen-specific T cells respond;

reintroducing the APCs into the patient, whereby auto-antigen-specific T cells are activated; and

administering a product which is detrimental to activated T cell proliferation in the patient.

42. The method of claim 41 wherein the polynucleotide which encodes all or a portion of an auto-antigen further encodes a signal sequence and a transmembrane/cytoplasmic tail, said signal sequence and transmembrane/cytoplasmic tail being functionally located with respect to the auto-antigen or portion thereof to facilitate the auto-antigen's endosomal processing.

43. The method of claim 41 wherein the product is Fas ligand.

44. The method of claim 43 wherein the Fas ligand is administered by administration of APC cells which express Fas ligand.

45. The method of claim 43 wherein the APC cells which express Fas ligand also express a truncated form of FADD, wherein said truncated form is sufficient to protect a cell which also expresses Fas from apoptosis.

46. The method of claim 45 wherein the APC cells which express FAS ligand and a truncated form of FADD are the same cells which express auto-antigen.

47. The method of claim 44 wherein the APC cells which express FAS ligand are the same cells which express auto-antigen.

48. The method of claim 41 wherein the polynucleotide is a recombinant viral genome.

49. The method of claim 48 wherein the viral genome encodes an attenuated virus.

50. The method of claim 48 wherein the viral genome is a Vaccinia virus genome.

51. The method of claim 48 wherein said polynucleotide further encodes the product which is detrimental to activated T cell proliferation.

52. The method of claim 51 wherein the product is Fas ligand.

53. The method of claim 52 wherein the polynucleotide further encodes a truncated form of FADD which is sufficient to protect a cell also expressing Fas from apoptosis.

54. Antigen presenting cells of an auto-immune disease patient which are transduced or transfected with (a) a first polynucleotide sequence encoding a protein comprising all or a portion of an auto-antigen to which the patient's antigen-specific T cells respond, said all or a portion of an auto-antigen being functionally connected to a signal peptide and a transmembrane/cytoplasmic tail, whereby endosomal processing of said all or a portion of the auto-antigen is facilitated, and (b) a second polynucleotide sequence which encodes a product detrimental to proliferation of activated T cells.

55. The antigen presenting cells of claim 54 wherein a single polynucleotide molecule comprises said first and second polynucleotide sequences.

56. The antigen presenting cells of claim 55 wherein the protein which is detrimental to activated T cell survival is Fas ligand.

57. The antigen presenting cells of claim 56 which have been transduced or transfected with a polynucleotide sequence encoding a truncated form of FADD which is sufficient to protect a cell also expressing Fas from apoptosis.

58. A virus which infects human APCs and which comprises (a) a first polynucleotide sequence which encodes all or a portion of an auto-antigen to which an auto-immune disease patient's antigen-specific T cells respond, and (b) a second polynucleotide sequence which encodes a product detrimental to proliferation of activated T cells.

59. The virus of claim 58 which is a Vaccinia virus.

60. The virus of claim 58, wherein the first polynucleotide sequence further encodes a signal sequence and a transmembrane/cytoplasmic tail both of which are functionally connected to said all or portion of the auto antigen, whereby the encoded all or a portion of the auto-antigen is processed by endosomes.

61. The virus of claim 60 wherein the auto-antigen is an extracellular domain of α-subunit of acetylcholine receptor and the auto-immune disease is *Myasthenia Gravis*.

62. The virus of claim 61 wherein the product is Fas ligand.

63. The virus of claim 62 further comprising a nucleotide sequence which

encodes a truncated form of FADD which is sufficient to protect a cell also expressing Fas from apoptosis.

64. The virus of claim 58 which is attenuated.

65. The method of claim 41 wherein the auto-antigen is an extracellular domain of α-subunit of acetylcholine receptor and the auto-immune disease is *Myasthenia Gravis*.

66. The antigen presenting cells of claim 54 wherein the auto-antigen is an extracellular domain of α-subunit of acetylcholine receptor and the auto-immune disease is *Myasthenia Gravis*.

67. The virus of claim 58 wherein the auto-antigen is an extracellular domain of α-subunit of acetylcholine receptor and the auto-immune disease is *Myasthenia Gravis*.

REMARKS

Claims 1-3, 6-8, 13, 15-18, 20, 21, 23-27, 29, 30, 32-34, and 36-40 are canceled without prejudice or disclaimer. Claims 4-5, 9-12, 14, 19, 22, 28, 31, and 35 are not currently under consideration, being drawn to non-elected species. Claims 41-67 have been added by amendment and are currently under consideration.

Support for claims 41-67 is found throughout the specification and in the originally filed claims. Indeed, claims 41-67, generally follow the format of the canceled claims. No new matter was introduced.

Support in the specification for claim 41 and for claims 65-67 is found, *inter alia*, at pages 5 to last paragraph of page 7, starting with first full paragraph on page 11 to end of first full paragraph on page 13, on page 14, and at the bottom of page 16 through first full paragraph on page 17. Support in the specification for claims 42, 54 and 60 is found,